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IDENTIFICATION OF NEW GENES FOR RESISTANCE TO TAN SPOT FOR USE IN DURUM AND COMMON WHEAT VARIETY IMPROVEMENT

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PROJECT NUMBER: 20000250-98BX

FINAL REPORT FOR PERIOD JULY 1, 2001 - DECEMBER 31, 2003

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ABSTRACT

Tan spot of wheat, caused by the fungus *Pyrenophora tritici-repentis,* is a wide-spread disease throughout Saskatchewan and can cause serious losses both in grain quality and yield. Symptoms of this disease are necrotic lesion and extensive chlorosis. This project was initiated to identify new sources of resistance to tan spot, to determine the genetic basis of this resistance and incorporate this resistance into registered durum and common wheat varieties. Plants were inoculated at the 2-leaf stage under controlled environmental conditions and resistance/susceptibility was assessed based on disease reaction. Resistance to tan spot was identified in a wide range of genetically diverse genotypes, including wild relatives of wheat. The F_1, F_2, and $F_{2:3}$ generations of each cross were tested. A single recessive gene was found to control resistance to necrosis caused by race 1 of *P. tritici-repentis* in all the resistant genotypes studied. The lack of segregation in the crosses between the resistant tetraploid and hexaploid genotypes when tested with race 1 indicated that they possess the same genes for resistance to necrosis and extensive chlorosis.

EXECUTIVE SUMMARY

Tan spot, a foliar disease of durum and common wheat, is caused by the fungus *Pyrenophora tritici-repentis* (Died.) Drechs. On average tan spot causes 5-15% yield losses and adversely affects the quality of grain by causing dark smudge and black point. The two effective means of controlling tan spot are use of genetic host resistance and use of fungicides. However, all durum and most common wheat varieties do not possess resistance. Application of fungicides is effective but is not cost-effective at current grain prices and creates environmental problems. Therefore, resistant varieties are the most effective and economical means of controlling tan spot.

Although resistance effective against the western Canadian races of *P. tritici-repentis* has been identified, the total number of genes currently available for resistance breeding is very small, e.g. only one gene for resistance to necrosis caused by races 1 and 2, the most common races in western Canada. The genetic base for breeding for tan spot resistant varieties is very narrow, emphasizing the

need to identify additional resistance genes to enable breeding resistant varieties with long-lasting and effective resistance.

The major objectives of this project were to i) identify new sources of resistance to the leaf disease tan spot of wheat and ii) determine through genetic studies if the resistance genes possessed by these resistant sources represent genes which have not been identified previously, and iii) incorporate the resistance into present grown wheat and durum varieties. In addition, the newly identified resistant sources were evaluated of resistance against leaf spot disease stagonospora nodorum blotch.

The wheat-tan spot pathosystem is believed to follow a gene-for-gene relationship with corresponding genes for resistance in the host and virulence in the pathogen. To ensure that the available host resistance genes are used effectively, plant breeders require knowledge of what races are present in the pathogen population of the areas for which they are developing resistant cultivars. Therefore, additional research was undertaken to determine the race composition of the Saskatchewan population of *P. tritici-repentis*.

A diverse range of wheat genotypes were tested under greenhouse conditions for disease reaction to different races of *P. tritici-repentis*. The material tested consisted of 429 genotypes of eight tetraploid species and one diploid wheat species, obtained from the USDA world wheat collection, and a 529 hexaploid wheat genotypes, including synthetic wheats, obtained from CIMMYT, Mexico and from other parts of the world.

Resistance to *P. tritici-repentis* races 1, 2, 3, and 5 for both the necrosis and chlorosis symptoms was identified in all ploidy levels of wheat, indicating the resistant sources may carry different resistance genes. More than 90% susceptible genotypes showed necrotic symptoms hence additional efforts should be made to breed for resistance to necrosis component of tan spot. Although majority of genotypes showed resistance to chlorosis, an equal proportion of accessions showed susceptibility to chlorosis induced by race 3 or 5. Therefore, screening for tan spot resistance should be based on all the virulent races of *P. tritici-repentis*.

To determine the inheritance of resistance to tan spot, crosses were made between newly identified resistant sources and susceptible genotypes. Crosses among the resistant genotypes were made to determine the allelic relationship between the gene(s) for resistance to tan spot. The F_1 and F_2 generations and $F_{2:3}$ families were tested with race 1, isolate Ptr D-2000-HT VI-5, at the 2-leaf stage under controlled environmental conditions and resistance/susceptibility was assessed seven days after inoculation based on lesion type.

Study of the genetic basis of resistance to necrosis induced by race 1 revealed that although the resistant parents used in this study were very genetically diverse, hexaploid synthetic wheat and representatives of four different species, *T. aestivum*, *T. durum*, *T. spelta* and *T. turgidum*, they all possessed the same single recessive gene for resistance. All the resistant sources also shared the gene for the chlorosis induced by race 1. These two genes, one for necrosis and one for chlorosis, were

common in both tetraploid and hexaploid wheat. Other studies have shown that the gene conferring resistance to necrosis caused by race 1 is located on chromosome 5B and that a major QTL for resistance to chlorosis caused by race 1 is located on chromosome 1A. Tetraploid and hexaploid wheats possess both A and B genomes and explains the occurrence of resistance. However, we observed resistance in genotypes that do not possess the B genome. Future studies should examine the genetic basis of resistance in these wild relatives of wheat. Such studies may identify novel resistant genes.

The resistant genotypes identified in this study showed resistance to both tan spot and stagonospora nodorum blotch. The genetic basis of resistance to stagonospora nodorum blotch needs to be determined. Studies done in our laboratory have shown that resistance to stagonospora nodorum blotch is controlled by a single recessive gene (Feng 2003). As the newly identified resistant sources possessed resistance to tan spot and stagonospora nodorum blotch, and the genetic basis is likely major genes in both cases, a study to determine if resistance to tan spot and stagonospora nodorum blotch is linked. This information would be very useful for plant breeders when planning their breeding strategies to develop cultivars resistant to these leaf spotting diseases.

The Saskatchewan population of *P. tritici-repentis* consisted of races 1, 2, 3, and 4 in the years 2000-2002. Throughout this study race 2 isolates were the most prevalent and were followed by race 1 on both durum and common wheat. Race 3 isolates were found only on durum wheat while race 4 isolates were found only on common wheat.

Although we observed only races 1-4 in our study this may not be the total composition of *P. tritici-repentis* population as this fungus infects different native grasses. Studies by others have identified six new races of *P. tritici-repentis* in the last three years. Most of the races were initially identified in areas where such surveys had not previously been conducted. However, these newly identified races were later found in other areas also. This indicates the need to conduct a comprehensive study in Saskatchewan of the *P. tritici-repentis* population obtained from common wheat, durum wheat and wild grasses. Full knowledge of the races present would indicate the appropriate resistance genes that should be used to develop varieties with effective and long-lasting resistance. Resistant reactions were observed by The barley, rye, oat and canaryseed genotypes used were resistant to all *P. tritici-repentis* isolates obtained for the race survey. However other studies have reported these species to be hosts of this pathogen. Therefore, further studies using optimized conditions for disease development should be conducted before before we can report with confidence that *P. tritici-repentis* is non-pathogenic on these crops.

The two resistant genes identified in these study are effective against the four races presently found in Saskatchewan. Recently races 5 and 11 were identified in the U.S.A. The newly identified sources of resistance in this study are effective against race 5, but it is not known if these sources are effective against race 11. This should be determined.

Almost all of the durum and bread wheat varieties currently registered for western Canada lack suitable resistance to tan spot, and thus producers look to fungicides for disease control until resistant varieties are available. However, continous use of fungicides may result in development of tolerant strains. It will be desirable to determine if the fungus *P. tritici-repentis* is capable of developing resistance or tolerance to the fungicides currently being used to determine the longivity of their effectiveness.

This study resulted in identification of genotypes showing resistance to all virulent races of *P. tritici-repentis* reported to occur in western Canada. Two genes controlling resistance to tan spot of wheat caused by race 1 were identified in this study. These two genes are effective against the races 1-4 of *P. tritici-repentis,* presently prevalent in Saskatchewan. The newly identified resistant genotypes show resistance to stagonospora nodorum blotch also. The resistance of these genotypes for tan spot and stagonospora nodorum blotch has been transferred into desired durum, CPS and HRS wheat background, which would facilitate development of leaf spot resistant varieties in near future.

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OBJECTIVES OF THE PROJECT

This project aims to 1) identify new sources of resistance to the leaf disease tan spot of wheat and 2) determine through genetic studies if the resistance genes possessed by these resistant sources represent genes which have not been identified previously, and can be used to broaden resources available for breeding resistant varieties.

1. IDENTIFICATION OF NEW SOURCES OF RESISTANCE TO TAN SPOT AND STAGNOSPORA NODORUM BLOTCH

GREENHOUSE SEEDLING TESTS

Materials

A diverse range of wheat genotypes have been tested in the greenhouse for disease reaction to *Pyrenophora tritici-repentis*, the fungus causing the leaf disease, tan spot of wheat. Materials used in these tests included a collection of wild diploid and tetraploid wheat, a collection of synthetic hexaploid and tetraploid wheat obtained from CIMMYT, Mexico, and tetraploid and hexaploid wheat genotypes collected from a variety of sources. A total of over 1,000 genotypes were tested at least once in greenhouse tests. Eight to ten plants per genotype were tested in each experiment.

Based on the initial disease screening, 40 genotypes showing good levels of resistance and coming from diverse genetic backgrounds were selected for further testing. These genotypes were tested for disease reaction to *Pyrenophora tritici-repentis* races 1, 2, 3, and 5 and for reaction to *Stagonospora nodorum*, cause of stagonospora nodorum blotch (syn. Septoria nodorum blotch). *P. tritici-repentis* race 1 isolates D-2000-HT VI-5 and W-2000-PA VII-9, race 2 isolates D-1998-WT VII-2 and W-2000-PA II-1, race 3 isolates D-2000-ST Vi-2 and D-2000-RT III-2, and race 5 isolates Ptr-DW 4 and Ptr-DW 13 were used in these tests. *Stagonospora nodorum* isolates Kelvington-SK, PRN-SK and Lang-SK were used to induce stagonospora nodorum blotch. Eight to ten plants per genotype were tested in each of the three replications of each test.

Disease Testing Procedures

Tan Spot of Wheat

Inoculum was produced using a modification of the method of Lamari and Bernier (1989). Mycelial plugs of 0.5-cm diameter from the stock cultures were transferred to 10-cm petri plates containing V8P agar (150 ml V8-juice, 10 g PDA, 10 g agar, 3 g $CaCO_3$ and 850 ml distilled water). These cultures were incubated in the dark at 20-22°C for six days. The plates were then flooded with sterile distilled water, the mycelium flattened with the base of a sterile test tube and the excess water poured off. To induce conidiophore production the plates were incubated under continuous light at room temperature for two days followed by one day in the dark in an incubator at 15-16°C to induce conidia production. The plates were flooded with distilled water and the conidia were suspended in the distilled water by gently brushing the mycelium with a camelhair brush to dislodge the conidia from the conidiophores. Spore concentration was measured with a haemocytometer and adjusted to 4,000 conidia per milliliter by addition of distilled water.

The pots were arranged at random before inoculation. Using a hand sprayer, plants at the two-leaf stage (approximately 14 days after seeding) were sprayed until runoff with the conidial suspension of the appropriate isolate. Following inoculation, the seedlings were incubated for 24 h in continuous leaf wetness in a mist chamber located in a growth room at 22/17°C (day/night) with a 16 h photoperiod and then returned to benches in the same growth room. Eight days after spore-inoculation, the seedlings were rated for disease reaction on a 1-5 lesion type rating scale developed by Lamari and Bernier (1989). A completely randomized design with three replications was used for each disease test.

Stagonospora Nodorum Blotch of Wheat

Inoculum was produced using a modified method of Ma (1993). Cultures were grown at room temperature on V8-agar medium (150 ml V8-juice, 15 g agar, 1.5 g $CaCO_3$ and 850 ml distilled water) in 10 cm diameter petri dishes under continuous illumination provided by 25-watt cool-white fluorescent tubes. New *S. nodorum* cultures were prepared by rubbing a 0.5 cm diameter plug of stock cultures with upside down on the surface of fresh V8-agar media. After seven days, pink pycnidia were obvious on the surface of V8-agar media and a spore suspension was prepared by flooding each petri dish with distilled water and gently brushing the medium surface with a camelhair brush to dislodge the spores. The resulting suspension was filtered through two layers of cheesecloth. The spore concentration was adjusted to 3.75×10^6 spores per milliliter by the addition of distilled water.

Seedlings were inoculated at the three-leaf stage (approx. 21 days after seeding) by spraying inoculum onto the leaves with a hand sprayer until runoff. The pots were arranged at random in the moist chamber prior to inoculation. After inoculation, the plants were kept in the moist chamber under continuous leaf wetness for 48 h with the same light and temperature regime as the growth room and then returned to the growth room benches. Seven days after inoculation, plants were rated for SNB lesion type using a 1-5 disease scale. This scale is based on the type, size and

number of lesions as well as the area of leaf covered by the chlorosis. A completely randomized design with three replications was also used for these disease tests.

RESULTS

Wild Diploid and Tetraploid Species

This collection, obtained from the USDA World Wheat Collection, represented eight species related to cultivated durum wheat and one diploid wheat species and was tested with isolate Ptr 200 (race 1) of *P. tritici-repentis*. Of the 429 accessions tested, 38 (9%) were highly resistant (rating 1) to tan spot (Table 1). Most belonged to either *T. timopheevii* or *T. monococcum*, but a small number occurred in three other tetraploid species, *T. dicoccoides*, *T. dicoccum* and *T. turgidum*. Overall, approximately 30% of the accessions were resistant (rating 1 or 2).

Table 1. Distribution of accession of each species for resistance to tan spot

Species	Genome	Number of accessions with disease reaction[a] of					Total	% Resistant
		1	2	3	4	5		
T. carthlicum	AB	0	6	13	30	1	50	12.0
T. dicoccoides	AB	2	6	6	1	1	16	50.0
T. dicoccum	AB	6	31	31	22	1	91	40.7
T. polonicum	AB	0	0	1	0	0	1	0.0
T. turanicum	AB	0	13	11	30	1	55	23.6
T. turgidum	AB	3	25	34	107	7	176	15.9
T. persicum	AB	0	0	1	0	0	1	0.0
T. timopheevii	AG	11	5	2	0	0	18	88.9
T. monococcum	A	16	4	0	1	0	21	95.2
Total		38	90	99	191	11	429	29.8

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

CIMMYT Synthetic Wheat

182 hexaploid synthetic lines and 35 tetraploid synthetic lines were tested with isolate Ptr 200 (race 1) of *P. tritici-repentis*. Based on the results of this test, 90 hexaploid and two tetraploid synthetic lines were identified as highly resistant and retested using an inoculum mixture containing races 1, 2, and 3. Those lines showing a high level of resistance to the race mixture seven days after inoculation were retained and rated again for disease reaction 14 days after inoculation. The best of those were then tested for resistance to stagonospora nodorum blotch (Table 2). Nine synthetics possessing the best combination of resistance to the two leaf spots were selected.

Table 2. Disease reaction[a] of synthetic wheat lines to tan spot and stagonospora nodorum blotch (SNB)

SN	Entry	Tan Spot			SNB	Remarks
		Ptr 200	Race Mixture			
		7 days	7 days	14 days	7 days	
85	SYN HEX Elite #60	1	1.6	2.5	1.5	
90	SYN HEX Elite #81	1	1.1	3.4	1.8	
92	SYN HEX Elite #9	1	1.1	1.4	1.0	Selected
94	SYN HEX Elite #85	1	1.4	1.5	1.0	Selected
100	SYN HEX Elite #24	1	1.4	2.3	1.4	
106	SYN HEX Elite #29	1	1.3	2.6	1.9	
107	SYN HEX Elite #73	1	1.1	1.9	1.8	
108	SYN HEX Elite #74	1	1.4	3.1	1.7	
114	SYN HEX Elite #54	1	1.1	2.6	2.2	
119	SYN HEX Elite #89	1	1.1	1.3	1.0	Selected
121	SYN HEX Elite #72	1	1.2	2.2	2.0	
125	SYN HEX Elite #25	1	1.2	1.3	1.0	Selected
126	SYN HEX Elite #67	1	1.4	1.7	1.0	Selected
127	SYN HEX Elite #1	1	1.2	1.4	1.0	Selected
129	SYN HEX Elite #3	1	1.3	1.7	1.3	
149	SYN HEX Elite #41	1	1.2	2.8	2.4	
150	SYN HEX Elite #42	1	1.1	2.9	2.1	
152	SYN HEX Elite #49	1	1.1	1.6	2.0	
157	SYN HEX Elite #53	1	1.2	1.2	1.0	
171	SYN HEX Elite #78	1	1.3	2.4	2.4	
232	ALTAR*$?Ae.	1	1.4	1.9	1.4	Selected
262	SH. Genome DD R	1	1.7	2.4	1.7	
267	Septoria Synthetics 47	1	1.6	2.4	1.5	
276	Septoria Synthetics 55	1	1.4	2.4	1.6	
277	Septoria Synthetics 56	1	1.5	3.1	2.0	
278	Septoria Synthetics 57	1	1.2	1.3	1.0	Selected
279	Septoria Synthetics 58	1	1.2	2.5	2.1	
280	Septoria Synthetics 59	1	1.2	1.4	1.2	
281	Septoria Synthetics 60	1	1.2	3.2	2.6	
282	Septoria Synthetics 61	1	1.4	3.7	3.4	
284	Septoria Synthetics 63	1	1.1	2.2	2.5	
289	Septoria Synthetics 67	1	1.3	2.5	2.1	
304	Septoria A Genome # 93	1	1.4	3.0	1.4	
318	Septoria A Genome # 106	1	1.4	1.4	1.0	Selected
	Erik (check)	1	1.6	2.1	1.5	Resistant
	Glenlea (check)	3.8	4.4	4.8	4.1	Susceptible
	Katepwa (check)	3.8	4.8	4.8	4.1	Susceptible

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Durum and Common Wheat Genotypes

312 genotypes in this group were tested. The level of tan spot resistance found was low and only 15 genotypes were selected for more extensive testing (Table 3).

Table 3. Disease reaction[a] of common wheat lines tested for resistance to tan spot and septoria nodorum blotch (SNB) in the seedling stage in greenhouse tests

SN #	Entry	Tan spot reaction			SNB	Remarks
		Ptr 200	Race mixture			
		7 days	7 days	14 days	7 Days	
245	LR Fusarium #1615	1.0	1.2	2.8	2.2	
329	SEPCIM60	1.1	1.4	1.5	1.2	
330	6SEPMON23	0.6	1.6	2.0	2.1	
340	SEPCIM46	0.6	1.4	2.2	1.6	
342	SEPCIM48	0.9	1.2	3.3	2.7	
343	SEPCIM47	1.2	1.1	1.7	1.7	
352	92MREHTR28B	1.1	1.4	1.7	0.8	Selected
434	CIMMYT LINES # 16	1.2	1.1	2.4	2.0	
436	CIMMYT LINE # 18	1.3	1.1	1.5	1.1	Selected
441	CIMMYT LINES # 17	1.4	1.1	1.8	2.0	
448	INTROS # 8	1.2	1.4	2.9	1.8	
453	CIMMYT LINES # 21	1.4	1.5	2.2	2.1	
457	INTROS # 7	1.3	1.3	1.9	1.4	Selected
489	MRHELSAT #55	1.3	1.5	2.6	1.7	
511	MRHELSAT #38	1.2	1.2	1.5	1.0	
	Erik (check)	1.3	1.6	2.1	1.5	Resistant
	Glenlea (check)	3.8	4.4	4.8	4.1	Susceptible
	Katepwa (check)	3.8	4.8	4.8	4.1	Susceptible

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Final Testing Phase

Genotypes SYN HEX Elite #1, SYN HEX Elite #9, SYN HEX Elite #25, SYN HEX Elite #67, SYN HEX Elite #85, SYN HEX Elite #89, Septoria Synthetics #57, Septoria A Gen Syn. #106, ALTAR*$?*Ae. squa.*, 92MREHTR28B, CIMMYT LINES #18, INTROS #7, 2000 Spelt #20, *T. timopheevii* (199), *T. dicoccoides* (206), *T. timopheevii* (227), *T. dicoccoides* (235), *T. turgidum* (283), *T. dicoccum* (420), *T. monococum* (429), and *T. monococum* (433) were further tested with each of races 1, 2, 3, and 5 of *P. tritici-repentis*, and a mixture of three isolates of *Stagonospora nodorum*. Each genotype showed a consistent resistant reaction to each of the *P. tritici-repentis* races and mixture of three isolates of *Stagonospora nodorum* used (Tables 4-8). Crosses were made with these resistant sources to create populations for determination of the genetic basis of their resistance.

Table 4. Disease reaction of genotypes screened in the seedling stage under controlled environmental conditions for resistance to *P. tritici-repentis* race 1

SN #	Genotype	*P. tritici-repentis* race 1 Reaction[a]				Symptoms Observed
		Rep 1	Rep 2	Rep 3	Mean	
Hexaploid Genotypes						
1	SYN HEX Elite #1	1.11	1.38	1.25	**1.25**	Resistant
2	SYN HEX Elite #9	1.00	1.33	1.11	**1.15**	Resistant
3	SYN HEX Elite #25	0.67	1.38	1.33	**1.13**	Resistant
4	SYN HEX Elite #67	1.33	1.22	1.13	**1.23**	Resistant
5	SYN HEX Elite #85	1.33	1.25	1.33	**1.31**	Resistant
6	SYN HEX Elite #89	1.25	1.11	1.13	**1.16**	Resistant
7	Septoria Synthetics 57	1.25	1.22	1.29	**1.25**	Resistant
8	Septoria A Gen Syn. 106	1.14	1.44	1.38	**1.32**	Resistant
9	ALTAR*$?*Ae. squa.*	2.00	1.63	1.67	**1.76**	Resistant
10	92MREHTR28B	1.22	1.50	1.38	**1.37**	Resistant
11	CIMMYT LINES # 18	1.33	1.56	1.38	**1.42**	Resistant
12	INTROS # 7	1.33	1.38	1.44	**1.38**	Resistant
13	2000 Spelt # 20	1.11	1.50	1.56	**1.39**	Resistant
14	Katepwa (check)	4.67	4.38	4.44	**4.50**	Necrosis
15	Glenlea (check)	4.56	4.25	4.38	**4.39**	Necrosis
16	Erik (check)	1.44	1.63	1.56	**1.54**	Resistant
17	Crocus	4.78	4.00	3.88	**4.22**	Necrosis
18	AC Splendor	4.11	4.50	3.89	**4.17**	Necrosis
19	CDC Teal	2.50	3.38	3.56	**3.14**	Necrosis
20	Kenyon (check)	4.67	4.56	4.56	**4.59**	Necrosis
21	6B-365 (check)	4.33	4.25	4.11	**4.23**	Chlorosis
22	6B-662 (check)	2.22	1.56	1.75	**1.84**	Necrosis
23	ND495 (check)	4.44	4.38	4.33	**4.38**	Necrosis
24	Janz	3.89	3.88	4.00	**3.92**	Necrosis
25	Domain	4.00	4.00	3.67	**3.89**	Necrosis
26	Conway	4.00	4.38	4.33	**4.24**	Necrosis
27	98W1147	3.57	3.89	3.67	**3.71**	Necrosis
28	Alsan	2.50	3.75	3.11	**3.12**	Necrosis
Tetraploid Genotypes						
29	Coulter (check)	4.44	4.11	4.50	**4.35**	Necrosis
30	AC Avonlea	2.33	4.38	3.78	**3.50**	Necrosis
31	4B-242 (check)	1.25	1.67	1.75	**1.56**	Resistant
32	4B-160	4.78	4.38	4.11	**4.42**	Nec+Chl
Wild Diploid (2n) and Tetraploid (4n) Accessions						
33	*T. timopheevii* (199) (4n)	1.29	1.25	1.33	**1.29**	Resistant
34	*T. dicoccoides* (206) (4n)	1.33	1.38	1.50	**1.40**	Resistant
35	*T. timopheevii* (227) (4n)	1.17	1.33	1.33	**1.28**	Resistant
36	*T. dicoccoides* (235) (4n)	1.33	1.11	1.25	**1.23**	Resistant
37	*T. turgidum* (283) (4n)	1.38	1.38	1.33	**1.36**	Resistant
38	*T. dicoccum* (420) (4n)	1.33	1.11	1.25	**1.23**	Resistant
39	*T. monococum* (429) (2n)	1.00	1.44	1.22	**1.22**	Resistant
40	*T. monococum* (433) (2n)	1.11	1.13	1.11	**1.12**	Resistant
	LSD (P=0.05)				**0.45**	

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Table 5. Disease reaction of genotypes screened in the seedling stage under controlled environmental conditions for resistance to *P. tritici-repentis* race 2

SN #	Genotype	*P. tritici-repentis* race 2 Reaction[a]				Symptoms Observed
		Rep 1	Rep 2	Rep 3	Mean	
Hexaploid Genotypes						
1	SYN HEX Elite #1	1.22	1.25	1.22	**1.23**	Resistant
2	SYN HEX Elite #9	1.29	1.13	1.25	**1.22**	Resistant
3	SYN HEX Elite #25	1.22	1.22	1.11	**1.19**	Resistant
4	SYN HEX Elite #67	1.11	1.11	1.29	**1.17**	Resistant
5	SYN HEX Elite #85	1.50	1.33	1.38	**1.40**	Resistant
6	SYN HEX Elite #89	1.14	1.13	1.33	**1.20**	Resistant
7	Septoria Synthetics 57	1.22	1.13	1.25	**1.20**	Resistant
8	Septoria A Gen Syn. 106	1.14	1.22	1.22	**1.20**	Resistant
9	ALTAR*$?*Ae. squa.*	1.33	1.25	1.33	**1.31**	Resistant
10	92MREHTR28B	1.22	1.38	1.38	**1.32**	Resistant
11	CIMMYT LINES # 18	1.22	1.25	1.29	**1.25**	Resistant
12	INTROS # 7	1.38	1.33	1.38	**1.36**	Resistant
13	2000 Spelt # 20	1.11	1.11	1.22	**1.15**	Resistant
14	Katepwa (check)	4.44	4.56	4.44	**4.48**	Necrosis
15	Glenlea (check)	4.00	4.25	4.33	**4.19**	Necrosis
16	Erik (check)	1.33	1.44	1.56	**1.44**	Resistant
17	Crocus	3.56	3.78	3.88	**3.74**	Necrosis
18	AC Splendor	2.88	3.00	3.11	**3.00**	Necrosis
19	CDC Teal	3.22	3.67	3.25	**3.38**	Necrosis
20	Kenyon (check)	4.11	4.38	4.33	**4.27**	Necrosis
21	6B-365 (check)	1.78	1.78	1.75	**1.77**	Resistant
22	6B-662 (check)	1.50	1.44	1.63	**1.52**	Resistant
23	ND495 (check)	3.13	3.44	3.67	**3.41**	Necrosis
24	Janz	2.88	3.38	3.33	**3.19**	Necrosis
25	Domain	3.75	3.33	3.56	**3.55**	Necrosis
26	Conway	3.50	4.33	4.00	**3.94**	Necrosis
27	98W1147	3.67	3.89	3.63	**3.73**	Necrosis
28	Alsan	3.11	3.44	3.33	**3.30**	Necrosis
Tetraploid Genotypes						
29	Coulter (check)	3.88	3.89	4.00	**3.92**	Necrosis
30	AC Avonlea	3.67	3.67	3.56	**3.63**	Necrosis
31	4B-242 (check)	1.33	1.38	1.56	**1.42**	Resistant
32	4B-160	1.80	1.56	1.78	**1.71**	Resistant
Wild Diploid (2n) and Tetraploid (4n) Accessions						
33	*T. timopheevii* (199) (4n)	1.13	1.13	1.22	**1.16**	Resistant
34	*T. dicoccoides* (206) (4n)	1.17	1.11	1.25	**1.18**	Resistant
35	*T. timopheevii* (227) (4n)	1.22	1.13	1.14	**1.16**	Resistant
36	*T. dicoccoides* (235) (4n)	1.13	1.11	1.22	**1.15**	Resistant
37	*T. turgidum* (283) (4n)	1.22	1.13	1.22	**1.19**	Resistant
38	*T. dicoccum* (420) (4n)	1.11	1.00	1.11	**1.07**	Resistant
39	*T. monococum* (429) (2n)	1.29	1.00	1.13	**1.14**	Resistant
40	*T. monococum* (433) (2n)	1.00	1.00	1.11	**1.04**	Resistant
	LSD (P=0.05)				**0.21**	

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Table 6. Disease reaction of genotypes screened in the seedling stage under controlled environmental conditions for resistance to *P. tritici-repentis* race 3

SN #	Genotype	*P. tritici-repentis* Race 3 Reaction[a]				Symptoms Observed
		Rep 1	Rep 2	Rep 3	Mean	
Hexaploid Genotypes						
1	SYN HEX Elite #1	1.33	1.44	1.38	**1.38**	Resistant
2	SYN HEX Elite #9	1.13	1.22	1.25	**1.20**	Resistant
3	SYN HEX Elite #25	1.44	1.33	1.38	**1.38**	Resistant
4	SYN HEX Elite #67	1.25	1.38	1.22	**1.28**	Resistant
5	SYN HEX Elite #85	1.22	1.33	1.25	**1.27**	Resistant
6	SYN HEX Elite #89	1.22	1.22	1.11	**1.19**	Resistant
7	Septoria Synthetics 57	1.22	1.25	1.13	**1.20**	Resistant
8	Septoria A Gen Syn. 106	1.25	1.38	1.22	**1.28**	Resistant
9	ALTAR*$?*Ae. squa.*	2.00	1.88	1.67	**1.85**	Resistant
10	92MREHTR28B	1.38	1.38	1.44	**1.40**	Resistant
11	CIMMYT LINES # 18	1.13	1.25	1.22	**1.20**	Resistant
12	INTROS # 7	1.25	1.38	1.33	**1.32**	Resistant
13	2000 Spelt # 20	1.22	1.11	1.22	**1.19**	Resistant
14	Katepwa (check)	1.89	2.00	2.13	**2.00**	Resistant
15	Glenlea (check)	1.89	2.00	2.11	**2.00**	Resistant
16	Erik (check)	1.33	1.38	1.44	**1.38**	Resistant
17	Crocus	2.00	1.89	2.00	**1.96**	Resistant
18	AC Splendor	2.25	2.33	2.33	**2.31**	Tip Chl.
19	CDC Teal	2.11	2.22	2.33	**2.22**	Tip Chl.
20	Kenyon (check)	2.38	2.44	2.56	**2.46**	Tip Chl.
21	6B-365 (check)	4.38	4.44	4.33	**4.38**	Chlorosis
22	6B-662 (check)	1.50	1.44	1.56	**1.50**	Resistant
23	ND495 (check)	1.78	1.89	2.00	**1.89**	Resistant
24	Janz	2.33	2.56	2.63	**2.50**	Tip Chl.
25	Domain	1.78	1.88	1.89	**1.85**	Resistant
26	Conway	2.22	2.44	2.56	**2.41**	Tip Chl.
27	98W1147	1.22	1.56	1.67	**1.48**	Resistant
28	Alsan	1.67	1.89	2.00	**1.85**	Resistant
Tetraploid Genotypes						
29	Coulter (check)	4.00	4.11	4.13	**4.08**	Necrosis
30	AC Avonlea	4.00	4.13	4.11	**4.08**	Necrosis
31	4B-242 (check)	1.67	1.44	1.50	**1.54**	Resistant
32	4B-160	4.00	4.11	4.13	**4.08**	Necrosis
Wild Diploid (2n) and Tetraploid (4n) Accessions						
33	*T. timopheevii* (199) (4n)	1.22	1.11	1.25	**1.19**	Resistant
34	*T. dicoccoides* (206) (4n)	1.25	1.13	1.11	**1.16**	Resistant
35	*T. timopheevii* (227) (4n)	1.33	1.13	1.25	**1.24**	Resistant
36	*T. dicoccoides* (235) (4n)	1.13	1.22	1.22	**1.19**	Resistant
37	*T. turgidum* (283) (4n)	1.44	1.44	1.33	**1.41**	Resistant
38	*T. dicoccum* (420) (4n)	1.44	1.33	1.38	**1.38**	Resistant
39	*T. monococum* (429) (2n)	1.56	1.44	1.33	**1.44**	Resistant
40	*T. monococum* (433) (2n)	1.33	1.38	1.50	**1.40**	Resistant
	LSD (P=0.05)				**0.15**	

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Table 7. Disease reaction of genotypes screened in the seedling stage under controlled environmental conditions for resistance to *P. tritici-repentis* race 5

SN #	Genotype	*P. tritici-repentis* Race 5 Reaction[a]				Symptoms Observed
		Rep 1	Rep 2	Rep 3	Mean	
Hexaploid Genotypes						
1	SYN HEX Elite #1	2.44	2.78	2.56	**2.59**	Chlorosis
2	SYN HEX Elite #9	3.22	3.22	2.89	**3.11**	Chlorosis
3	SYN HEX Elite #25	3.00	3.13	2.67	**2.93**	Chlorosis
4	SYN HEX Elite #67	1.11	1.22	1.44	**1.26**	Resistant
5	SYN HEX Elite #85	1.22	1.25	1.50	**1.32**	Resistant
6	SYN HEX Elite #89	1.00	1.11	1.13	**1.08**	Resistant
7	Septoria Synthetics 57	1.11	1.13	1.22	**1.15**	Resistant
8	Septoria A Gen Syn. 106	1.00	1.11	1.13	**1.08**	Resistant
9	ALTAR*$?*Ae. squa.*	1.11	1.33	1.22	**1.22**	Resistant
10	92MREHTR28B	1.11	1.25	1.11	**1.16**	Resistant
11	CIMMYT LINES # 18	1.00	1.11	1.22	**1.11**	Resistant
12	INTROS # 7	1.11	1.22	1.25	**1.19**	Resistant
13	2000 Spelt # 20	1.00	1.50	1.22	**1.24**	Resistant
14	Katepwa (check)	3.88	4.11	4.22	**4.07**	Chlorosis
15	Glenlea (check)	1.50	1.67	1.56	**1.57**	Resistant
16	Erik (check)	1.22	1.33	1.33	**1.30**	Resistant
17	Crocus	1.38	1.44	1.56	**1.46**	Resistant
18	AC Splendor	2.29	2.50	2.67	**2.48**	Chlorosis
19	CDC Teal	1.67	2.11	2.13	**1.97**	Resistant
20	Kenyon (check)	3.75	3.71	3.88	**3.78**	Chlorosis
21	6B-365 (check)	2.13	1.89	2.11	**2.04**	Resistant
22	6B-662 (check)	3.63	3.78	3.89	**3.76**	Chlorosis
23	ND495 (check)	2.00	2.11	2.22	**2.11**	Resistant
24	Janz	2.00	2.22	2.33	**2.19**	Tip Chl.
25	Domain	4.44	3.89	3.44	**3.93**	Chlorosis
26	Conway	4.00	4.00	4.22	**4.07**	Chlorosis
27	98W1147	1.11	1.33	1.25	**1.23**	Resistant
28	Alsan	1.44	1.67	1.56	**1.56**	Resistant
Tetraploid Genotypes						
29	Coulter (check)	4.00	4.22	4.25	**4.16**	Necrosis
30	AC Avonlea	2.25	2.22	2.56	**2.34**	Necrosis
31	4B-242 (check)	1.44	1.56	1.63	**1.54**	Resistant
32	4B-160	2.38	2.22	2.33	**2.31**	Necrosis
Wild Diploid (2n) and Tetraploid (4n) Accessions						
33	*T. timopheevii* (199) (4n)	1.38	1.13	1.25	**1.25**	Resistant
34	*T. dicoccoides* (206) (4n)	1.25	1.00	1.38	**1.21**	Resistant
35	*T. timopheevii* (227) (4n)	1.00	1.11	1.13	**1.08**	Resistant
36	*T. dicoccoides* (235) (4n)	1.38	1.22	1.33	**1.31**	Resistant
37	*T. turgidum* (283) (4n)	1.67	1.33	1.38	**1.46**	Resistant
38	*T. dicoccum* (420) (4n)	1.13	1.11	1.25	**1.16**	Resistant
39	*T. monococum* (429) (2n)	1.00	1.25	1.22	**1.16**	Resistant
40	*T. monococum* (433) (2n)	1.00	1.44	1.11	**1.19**	Resistant
	LSD (P=0.05)				**0.25**	

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

Table 8. Disease reaction of genotypes screened in the seedling stage under controlled environmental conditions for resistance to stagonospora nodorum blotch

SN #	Genotype	Disease reaction[a]				Resistance evaluation
		Rep 1	Rep 2	Rep 3	Mean	
Hexaploid and Tetraploid Genotypes						
1	SYN HEX Elite #1	1.00	1.11	1.13	**1.08**	Resistant
2	SYN HEX Elite #9	1.14	1.00	1.11	**1.08**	Resistant
3	SYN HEX Elite #25	1.13	1.13	1.22	**1.16**	Resistant
4	SYN HEX Elite #67	1.11	1.00	1.14	**1.08**	Resistant
5	SYN HEX Elite #85	1.00	1.13	1.22	**1.12**	Resistant
6	SYN HEX Elite #89	1.00	1.11	1.13	**1.08**	Resistant
7	Septoria Synthetics 57	1.11	1.00	1.11	**1.07**	Resistant
8	Septoria A Gen Syn. 106	1.00	1.22	1.25	**1.16**	Resistant
9	ALTAR*$?*Ae. squa.*	1.00	1.13	1.33	**1.15**	Resistant
10	92MREHTR28B	1.13	1.11	1.22	**1.15**	Resistant
11	CIMMYT LINES # 18	2.00	1.11	1.25	**1.45**	Resistant
12	INTROS # 7	1.88	1.22	1.13	**1.41**	Resistant
13	2000 Spelt # 20	1.13	1.00	1.11	**1.08**	Resistant
14	Katepwa (check)	4.44	4.44	4.38	**4.42**	Susceptible
15	Glenlea (check)	3.22	3.63	3.33	**3.39**	Susceptible
16	Erik (check)	1.11	1.22	1.25	**1.19**	Resistant
17	Crocus	3.50	3.00	3.44	**3.31**	Susceptible
18	AC Splendor	3.25	2.78	3.13	**3.05**	Susceptible
19	CDC Teal	4.29	4.25	4.11	**4.22**	Susceptible
20	Kenyon (check)	4.50	4.22	4.25	**4.32**	Susceptible
21	6B-365 (check)	4.25	4.11	3.56	**3.97**	Susceptible
22	6B-662 (check)	3.78	2.33	2.89	**3.00**	Susceptible
23	ND495 (check)	4.11	3.56	4.25	**3.97**	Susceptible
24	Janz	3.50	3.67	3.63	**3.60**	Susceptible
25	Domain	4.44	4.22	4.13	**4.26**	Susceptible
26	Conway	4.11	3.78	3.88	**3.92**	Susceptible
27	98W1147	2.13	2.75	2.25	**2.38**	Susceptible
28	Alsan	4.14	3.22	3.33	**3.57**	Susceptible
29	Coulter (check)	3.88	4.11	4.13	**4.04**	Susceptible
30	AC Avonlea	1.38	1.33	1.67	**1.46**	Resistant
31	4B-242 (check)	2.25	2.33	2.22	**2.27**	Susceptible
32	4B-160	3.11	3.43	3.13	**3.22**	Susceptible
Wild Diploid (2n) and Tetraploid (4n) Accessions						
33	*T. timopheevii* (199) (4n)	1.11	1.11	1.22	**1.15**	Resistant
34	*T. dicoccoides* (206) (4n)	1.13	1.22	1.33	**1.23**	Resistant
35	*T. timopheevii* (227) (4n)	1.22	1.00	1.25	**1.16**	Resistant
36	*T. dicoccoides* (235) (4n)	1.11	1.11	1.22	**1.15**	Resistant
37	*T. turgidum* (283) (4n)	1.11	1.14	1.25	**1.17**	Resistant
38	*T. dicoccum* (420) (4n)	1.14	1.00	1.11	**1.08**	Resistant
39	*T. monococum* (429) (2n)	1.17	1.13	1.13	**1.14**	Resistant
40	*T. monococum* (433) (2n)	1.13	1.00	1.11	**1.08**	Resistant
	LSD (P=0.05)				**0.36**	

[a] Based on a 1-5 scale, where 1 = highly resistant, 5 = susceptible.

DISCUSSION

This study showed that resistance to tan spot is present in a wide range of genetically diverse genotypes, including certain wild relatives of wheat. This agrees with previous reports (Lamari and Bernier 1989) and suggests that a number of different genes for resistance may be present in the primary and secondary gene pools of wheat. Resistance was found in all ploidy levels indicating that the A genome must carry at least one gene for resistance. Among the wild relatives tested, *T. monococcum* and *T. timopheevii* had the highest number of highly resistant accessions, whereas none were in other species such as *T. carthlicum* and *T. turanicum*. This indicates that not all wild wheat relatives are likely to be useful sources of resistance.

One-half of the hexaploid synthetic wheats tested were highly resistant. Since the wild relative *Aegilops squarrosa* is a common parent in all these wheats, this suggests that this species, believed the contributor of the D genome in common wheat, may also be a valuable source of resistance to tan spot.

This study included a number of wheat cultivars registered in North America. Only the US spring wheat Erik possessed resistance to tan necrosis, however resistance to the extensive chlorosis symptom was more common. Erik and Alsan, also a US spring wheat, and the Canadian cultivar Glenlea were resistant, while AC Domain, AC Splendor and CDC Teal showed moderate resistance.

FIELD DISEASE NURSERIES

All synthetic wheat, durum and common wheat genotypes were grown in replicated short rows at the North Seed Farm, Saskatoon in 2001 for field evaluation of tan spot reaction. However, despite repeated inoculation of this nursery and irrigation, a disease epidemic could not be established because of the extremely dry conditions experienced during the summer. No data was obtained from this nursery.

All 40 genotypes tested under controlled environmental conditions and selected for further study were planted in replicated short row nurseries at North Seed Farm, Saskatoon in 2002 and 2003 for field evaluation of tan spot and stagonospora nodorum blotch reaction. The nursery was seeded at two different dates with the objective of evaluating disease reaction at both the seedling and the adult plant stage. Disease inoculation involved both use of infected straw and spore inoculation. However, the extreme dry weather conditions experienced in both years led to failure for disease to develop sufficiently for meaningful data to be obtained. Therefore, as experienced in 2001, no data was obtained from this nursery.

However, supplementary irrigation enabled the nursery to serve as a seed increase for the 40 genotypes selected in the seedling tests. A limited amount of seed of these genotypes is available for use by other researchers upon request.

$F_{2:4}$ families of crosses segregating for leaf spot resistance were grown in 2003 in a second field nursery to be screened for resistance to tan spot and stagonospora nodorum blotch. However, for reasons explained above, this nursery also failed, but did provide the opportunity to advance these populations to the F_5 generation.

2. PRODUCTION OF POPULATIONS FOR GENETIC STUDIES

A total of 143 crosses were made among the different resistant and susceptible genotypes selected for this study. These crosses were made to enable study of the genetic control of resistance and to determine the allelic relationships among the resistance genes.

While most crosses produced F_1 seed, hybrid necrosis was observed in the F_1 plants of certain crosses with synthetic wheat as one of the parents. This resulted in failure of F_2 seed to be obtained for those crosses. Embryo rescue was used to rescue the F_1 embryos produced in crosses involving wild relatives of wheat. However, with the exception of the crosses involving *T. turgidum* # 283, all F_1 plants of the crosses involving wild relatives as one parent were both male- and female-sterile. Plant growth of these embryo-rescued plants was normal, however they failed to set seed. Backcrosses to these F_1 plants was attempted but only in three crosses, Crocus x *T. timopheevii* (199) x Crocus, Crocus x *T. dicoccoides* (235) x Crocus and Crocus x *T. dicoccoides* (206) x Crocus, were a few BC_1F_1 embryos obtained.

Due to space limitations or unavailability of F_2 embryos, advanced generation populations for only 26 cross populations were produced (Table 9). For each of these crosses approximately 110-125 $F_{2:3}$ families were produced. These families were advanced to the F_4 and then to the F_5 generation in either the greenhouse or the field.

Details of the other crosses made are presented in Appendix Tables I-II.

GENETICS OF RESISTANCE TO TAN SPOT OF WHEAT

Isolates of *P. tritici-repentis* are classified into 11 races based on their ability to induce necrosis and/or chlorosis on a set of wheat differential cultivars (Ali and Francl 2002, Ali et al. 2002; Manning et al. 2002; Lamari et al. 2003). Races 1-5 have been found in the U.S.A., while studies done in our laboratory have shown that the Saskatchewan population of *P. tritici-repentis* is composed of races 1, 2, 3, and 4 (see Section 4 of this report). Race 1 possesses the virulence of both race 2 and race 3, while race 4 is avirulent. Genetic studies with race 1 will identify the resistance genes currently required in western Canadian wheat varieties to provide resistance to tan spot. Therefore, race 1 of *P. tritici-repentis* was used in this study to determine the genetic control of resistance to tan spot.

Table 9. Listing of the crosses made and the advanced generation populations produced

Cross #	Cross	F_1's	F_2 Embryos	$F_{2:3}$/$F_{2:4}$/ $F_{2:5}$ Families
4	Syn. Hex. Elite # 1 x 98W1147	50	200+ embryos	40 DH Lines
8	Syn. Hex. Elite # 9 x 98W1147	29	200+ embryos	110/87/81 families
12	Syn. Hex. Elite # 25 x 98W1147	48	200+ embryos	110/86/83 families
16	Syn. Hex. Elite # 67 x 98W1147	24	100+ embryos	120/88/81 families
20	Syn. Hex. Elite # 85 x 98W1147	27	200+ embryos	120/100/97 families
26	Syn. Hex. Elite # 89 x 98W1147	22	200+ embryos	120/93/86 families
29	Septoria Syn. # 57 x Kenyon	9	200+ embryos	120/114/112 families
39	92MREHTR28B x Erik	41	200+ embryos	110/83/80 families
40	92MREHTR28B x Kenyon	59	200+ embryos	110/80/80 families
44	CIMMYT LINE # 18 x Erik	19	200+ embryos	110/86/85 families
45	CIMMYT LINE # 18 x Kenyon	31	200+ embryos	110/81/81 families
51	INTROS # 7 x Erik	10	100+ embryos	110/94/87 families
52	INTROS # 7 x Kenyon	12	100+ embryos	110/102/96 families
56	2000 Spelt # 20 x Erik	16	200+ embryos	110/86/83 families
57	2000 Spelt # 20 x Kenyon	16	200+ embryos	110/98/97 families
63	Altar*S/*Ae. squa.* x Erik	8	100+ embryos	120/96/87 families
64	Altar*S/*Ae. squa.* x Kenyon	9	200+ embryos	120/111/100 families
113	*T. turgidum* (283) x Crocus	16	200+ embryos	125 $F_{2:3}$ families
114	*T. turgidum* (283) x Katepwa	29	200+ embryos	Yes
115	*T. turgidum* (283) x Erik	8	200+ embryos	85 $F_{2:3}$ families
116	*T. turgidum* (283) x 98W1147	52	200+ embryos	90 $F_{2:3}$ families
118	*T. turgidum* (283) x Coulter	10	200+ embryos	120/110/84 families
119	Coulter x Katepwa	16	100+ embryos	50 $F_{2:3}$ families
124	Katepwa x Coulter	10	100+ embryos	50 $F_{2:3}$ families
129	Katepwa x *T. turgidum* (283)	2	200+ embryos	55 $F_{2:3}$ families
137	Erik x 4B-242	38	200+ embryos	30 $F_{2:3}$ families

OBJECTIVES

The objectives of this component of the project were (1) to determine the genetic control resistance to tan spot in newly identified sources of resistance, and (2) to determine if the resistance genes possessed by these sources represented genes which had not been identified previously.

MATERIAL AND METHODS

The F_1 and F_2 generations and the $F_{2:3}$ families of 12 crosses were screened with race 1, isolate Ptr D-2000-HT VI-5 (Table 10). A family size of 16-18 plants was used, which gives a 99% probability of distinguishing between a segregating and a non-segregating family. Inoculum production, inoculation and disease rating was done using the methods described earlier. Plants were rated on a 1-5 lesion type rating

scale with plants rating 1 or 2 being considered resistant, while those with ratings of 3 to 5 were considered susceptible.

RESULTS

Genetic Control of Resistance

All 12 crosses studied were observed to segregate for the tan necrosis component of the tan spot symptoms caused by race 1, but not for the extensive chlorosis component. This indicated that all parents used in these crosses were resistant to chlorosis produced by race 1. The F_1 plants of all resistant x susceptible crosses involving hexaploid parents were susceptible, indicating that resistance to necrosis was recessive (Table 10).

The F_2 generations of these crosses segregated in a 1 resistant: 3 susceptible ratio, indicating single gene control of resistance and confirming that resistance to necrosis was recessive (Table 10). The $F_{2:3}$ families segregated in the ratio 1 homozygous resistant: 2 segregating: 1 homozygous susceptible, thus supporting the hypothesis of monogenic control of resistance to necrosis (Table 11). For cross SHE # 1 x 98W1147, a doubled haploid (DH) population was developed and 45 DH lines were produced. These DH lines segregated in a 1 resistant: 1 susceptible ratio, confirming single gene control in this cross. The segregation data for the resistant x susceptible tetraploid wheat cross (*T. turgidum* # 283 x Coulter) confirmed that resistance to necrosis in this cross was also controlled by single gene. In addition, a single gene was found to control resistance to necrosis in the resistant (tetraploid) x susceptible (hexaploid) cross, *T. turgidum* # 283 x 98W1147 (Table 11).

Allelism Studies

Since all parents of the crosses studied were resistant to chlorosis caused by race 1 and no segregation for resistance to chlorosis was observed, it was concluded that the resistance sources from tetraploid and hexaploid wheat possessed same gene for resistance to chlorosis.

All F_1 plants of the crosses of five resistant hexaploid genotypes with the resistant cultivar Erik were resistant to race 1 (Table 10). However, while most F_2 plants of these crosses were resistant, a few plants in four of the crosses gave a moderately susceptible reaction (rating 3). However, all of the $F_{2:3}$ families of these crosses were homozygous resistant, thus confirming that all six resistant parents possessed the same resistance gene (Table 11). The cross between resistant tetraploid genotype *T. turgidum* # 283 and resistant hexaploid cultivar Erik did not segregate for resistance (Tables 10, 11). This indicated that the tetraploid resistant genotype and the hexaploid resistant genotypes studied possessed the same single gene for resistance to necrosis produced by race 1 of *P. tritici-repentis*.

Table 10. F_1 plant disease reaction to isolate Ptr D-2000-HT VI-5 and chi-square tests of F_2 segregation ratios of crosses used to study the inheritance and allelic relationships among the gene(s) controlling resistance to tan spot caused by race 1

Cross		Number of plants				Ratio tested	χ^2 value	P value[c]
SN #	Pedigree	F_1 gen. R[a]	F_1 gen. S[b]	F_2 gen. R	F_2 gen. S			
Resistant (Hexaploid) x Susceptible (Hexaploid)								
4	SHE # 1 x 98W1147	0	7	71	173	1:3	2.19	0.14
8	SHE # 9 x 98W1147	0	9	55	173	1:3	0.94	0.76
12	SHE # 25 x 98W1147	0	8	79	190	1:3	2.74	0.09
16	SHE # 67 x 98W1147	0	9	72	166	1:3	3.50	0.06
20	SHE # 85 x 98W1147	0	7	76	180	1:3	3.00	0.08
26	SHE # 89 x 98W1147	0	7	52	124	1:3	1.94	0.16
29	Sep. Syn. # 57 x Kenyon	0	8	75	175	1:3	3.33	0.07
57	2000 Spelt # 20 x Kenyon	0	7	74	177	1:3	2.69	0.10
52	Intros # 7 x Kenyon	0	8	60	196	1:3	0.33	0.56
45	CIMMYT L # 18 x Kenyon	0	9	61	198	1:3	0.29	0.59
64	Altar*S/*Ae. squ.* x Kenyon	0	8	54	202	1:3	2.08	0.14
40	92MREHTR28B x Kenyon	0	7	60	197	1:3	0.37	0.54
Resistant (Tetraploid) x Susceptible (Tetraploid)								
118	*T. turgidum* # 283 x Coulter	0	7	74	181	1:3	2.19	0.13
Resistant (Tetraploid) x Susceptible (Hexaploid)								
116	*T. turgidum* #283 x 98W1147	0	7	20	54	1:3	0.16	0.68
Resistant (Hexaploid) x Resistant (Hexaploid)								
56	2000 Spelt #20 x Erik	8	0	178	2	1:0	-	-
51	Intros #7 x Erik	9	0	256	0	1:0	-	-
44	CIMMYT Line #18 x Erik	8	0	248	8	1:0	-	-
63	Altar*S/*Ae. squ.* x Erik	9	0	255	1	1:0	-	-
39	92MREHTR28B x Erik	7	0	251	5	1:0	-	-
Resistant (Tetraploid) x Resistant (Hexaploid)								
115	*T. turgidum* # 283 x Erik	7	0	192	0	1:0	-	-

[a] Plants showing resistant reaction. [b] Plants showing susceptible reaction.
[c] Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that the observed population does not differ significantly from expected hypothesis.

Table 11. Chi-square test for $F_{2:3}$ family segregation ratios of crosses used to study the inheritance and allelic relationships for resistance to tan spot caused by Ptr D-2000-HT VI-5 (race 1)

Cross		Number of $F_{2:3}$ families			Ratio tested	χ^2 value	P value[b]
SN #	Pedigree	Res.	Seg.[a]	Sus.			
Resistant (Hexaploid) x Susceptible (Hexaploid)							
4	SHE #1 x 98W1147	23	-	22	1:1	0.02	0.89
8	SHE #9 x 98W1147	17	45	18	1:2:1	1.18	0.56
12	SHE #25 x 98W1147	22	45	13	1:2:1	2.05	0.36
16	SHE #67 x 98W1147	22	44	14	1:2:1	1.50	0.47
20	SHE #85 x 98W1147	14	47	19	1:2:1	3.05	0.22
26	SHE #89 x 98W1147	17	45	18	1:2:1	1.18	0.56
29	Sep. Syn. #57 x Kenyon	22	40	18	1:2:1	0.30	0.86
57	2000 Spelt #20 x Kenyon	15	45	20	1:2:1	1.87	0.39
52	Intros # 7 x Kenyon	14	44	22	1:2:1	1.50	0.47
45	CIMMYT Line #18 x Kenyon	14	44	22	1:2:1	1.50	0.47
64	Altar*S/*Ae. squ.* x Kenyon	13	42	25	1:2:1	3.13	0.21
40	92MREHTR28B x Kenyon	14	45	21	1:2:1	2.45	0.29
Resistant (Tetraploid) x Susceptible (Tetraploid)							
118	*T. turgidum* #283 x Coulter	17	33	20	1:2:1	0.36	0.86
Resistant (Tetraploid) x Susceptible (Hexaploid)							
116	*T. turgidum* #283 x 98W1147	20	36	14	1:2:1	0.74	0.69
Resistant (Hexaploid) x Resistant (Hexaploid)							
56	2000 Spelt #20 x Erik	80	0	0	1:0:0	-	-
51	Intros #7 x Erik	80	0	0	1:0:0	-	-
44	CIMMYT Line #18 x Erik	80	0	0	1:0:0	-	-
63	Altar*S/*Ae. Squ.* x Erik	80	0	0	1:0:0	-	-
39	92MREHTR28B x Erik	80	0	0	1:0:0	-	-
Resistant (Tetraploid) x Resistant (Hexaploid)							
115	*T. turgidum* #283 x Erik	70	0	0	1:0:0	-	-

[a] Segregating for resistance.

[b] Probability of obtaining deviations from the expected ratio by chance alone. A probability value greater than 0.05 indicates that segregation in the observed population does not differ significantly from the expected ratio.

DISCUSSION

This study showed that seedling resistance to the tan necrosis caused by *P. tritici-repentis* was controlled by a single recessive gene in each of the durum and common wheat crosses studied. This finding agrees with earlier studies with different sources of resistance by Lamari and Bernier (1991) and Singh (2001). Although resistance to extensive chlorosis was not studied, the absence of segregation for chlorosis in the crosses studied indicates that all the resistance sources possessed the same gene for resistance to extensive chlorosis caused by race 1.

Since the resistant parents used in this study were very genetically diverse, hexaploid synthetic wheat and representatives of four different species, *T. aestivum*, *T. durum*, *T. spelta* and *T. turgidum*, the finding that all these new resistance sources possessed the same gene controlling resistance to tan necrosis caused by race 1 was unexpected. Singh (2001) found that resistance in each of a different set of resistant genotypes was controlled by the same resistance gene as in Erik. Other workers (Gamba et al. 1998; Duguid and Brûlé-Babel 2001) have also reported that resistance to tan necrosis was due to a single gene common to all the resistant genotypes studied. Although not all of these studies contained cv. Erik, these findings suggest very strongly that the wheat gene pool possesses little genetic diversity for resistance to the tan necrosis component of tan spot. Stock et al. (1996) and Anderson et al. (1999) reported that this gene (*tsn1*) is located on chromosome 5BL in both durum and hexaploid wheat. In Section 1 of this project a number of *T. monococcum* accessions were found to be highly resistant to tan necrosis. Since *T. monococcum* carries only the A genome, this genome carries resistance under different gene control than that reported above. This is supported by Effertz et al. (2001) who identified a major QTL for resistance to the race 1 toxin causing extensive chlorosis on chromosome 1AS. However, it does appear that the genetic base for resistance to tan spot is narrow and that efforts should continue to seek new and different genes for resistance.

3. TRANSFER OF TAN SPOT RESISTANCE INTO ADAPTED DURUM, HARD RED SPRING, AND CANADIAN PRAIRIE SPRING WHEAT GERMPLASM

Breeding populations to produce leaf spot resistant parental material adapted to western Canada are being developed through backcrossing and production of three-way crosses involving advanced breeding lines and registered cultivars.

The backcross populations produced are shown in Table 12. The backcross program used the adapted susceptible parent as the recurrent parent to produce BC_1F_1 embryos which were grown in the greenhouse to produce BC_1F_2 families which were screened at the seedling stage for resistance to both tan spot and stagnospora nodorum blotch. Plants resistant to both diseases were identified and backcrossed to produce BC_2F_1 embryos. The resulting BC_2F_1 plants were grown to produce BC_2F_2 embryos.

Table 12. Details of backcross breeding populations produced to transfer resistance to tan spot and stagonospora nodorum blotch into CPS and CWRS wheat germplasm

Cross #	Cross pedigree	BC_1F_1 embryos	BC_1F_2 families	BC_1F_3 embryos	BC_2F_1 embryos	BC_2F_2 embryos
1	Spelt #20 x Kenyon	Available	57	1000+	Available	500+
2	Spelt #20 x 98W1147	Available	42	1000+	Available	500+
3	Spelt #20 x CDC Teal	Available	48	1000+	Available	500+
4	Intros # 7 x Kenyon	Available	45	1000+	Available	500+
5	Intros # 7 x 98W1147	Available	46	1000+	Available	500+
6	Intros # 7 x CDC Teal	Available	48	1000+	Available	500+

Two durum three-way (cross #s 26, 27), 9 CPS three-way (cross #s 3, 4, 7, 8, 9, 13, 17, 21, 25), and 16 HRS three-way (cross #s 1, 2, 5, 6, 10, 11, 12, 14, 15, 16, 18, 19, 20, 22, 23, 24) cross populations have been produced involving recent and current registered cultivars/lines. In crosses #1-8, BC_1F_2 plants resistant to tan spot and stagnospora nodorum blotch were crossed with selected CPS and HRS wheat cultivars and F_2 embryos produced. For crosses #9-27, $F_{2:3}$ families of each single cross were screened for tan spot and stagnospora nodorum blotch resistance and plants from these families were crossed with agronomically superior durum, CPS, and HRS wheat cultivars. The U.S. cultivar Alsan was also used as a parent to introduce fusarium head blight resistance. F_2 embryos of all crosses have been produced. Selections from these breeding populations will be used as parental material in the CWRS, CPS and durum breeding programs of the Crop Development Centre, University of Saskatchewan.

4. RACE STRUCTURE OF THE *PYRENOPHORA TRITICI-REPENTIS* POPULATION IN SASKATCHEWAN

Different races of *P. tritici-repentis* produce different combinations of the two components of the tan spot symptoms, tan necrosis and extensive chlorosis, and resistance to each component is controlled by different genes. Therefore, knowledge of the race structure of the tan spot fungus is important so that the appropriate races can be used in disease testing to ensure that the correct resistance genes are being selected for when breeding resistant cultivars.

To date, 11 races have been identified throughout the world (Ali and Francl 2002, Ali et al. 2002; Manning et al. 2002; Lamari et al. 2003), although only six have been found in North America. However, only limited information is available on the races occurring in Manitoba and nothing is known of the current race makeup of the Saskatchewan population of *P. tritici-repentis*. Since the full value of this research project cannot be realized without this information, this study to identify the races occurring in Saskatchewan was undertaken. This study was not part of the original work plan for this project.

Table 13. Details of the three-way breeding populations developed to transfer resistance to tan spot and stagonospora nodorum blotch into durum, CPS and CWRS wheat varieties.

Cross #	Cross pedigree	F_1 Embryos	F_2 Embryos
1	(Spelt # 20 x Kenyon* 2) x Alsan	Available	500+
2	(Spelt # 20 x CDC Teal* 2) x Alsan	Available	500+
3	(Spelt # 20 x 98W1147*2) x HY 475	Available	500+
4	(Spelt # 20 x 98W1147*2) x HY 644	Available	500+
5	(INTROS # 7 x Kenyon* 2) x Alsan	Available	500+
6	(INTROS # 7 x CDC Teal* 2) x Alsan	Available	500+
7	(INTROS # 7 x 98W1147*2) x HY 475	Available	500+
8	(INTROS # 7 x 98W1147*2) x HY 644	Available	500+
9	(Altar*S/Ae.squ. x Kenyon) x HY 644	Available	1000+
10	(Altar*S/Ae.squ. x Kenyon) x Alsan	Available	1000+
11	(Altar*S/Ae.squ. x Kenyon) x AC Splendor	Available	1000+
12	(Altar*S/Ae.squ. x Kenyon) x CDC Bounty	Available	1000+
13	(92 MREHTR 28B x Kenyon) x HY 644	Available	1000+
14	(92 MREHTR 28B x Kenyon) x Alsan	Available	1000+
15	(92 MREHTR 28B x Kenyon) x AC Splendor	Available	1000+
16	(92 MREHTR 28B x Kenyon) x CDC Bounty	Available	1000+
17	(Sep. Syn.# 57 x Kenyon) x HY 644	Available	1000+
18	(Sep. Syn.# 57 x Kenyon) x Alsan	Available	1000+
19	(Sep. Syn.# 57 x Kenyon) x AC Splendor	Available	1000+
20	(Sep. Syn.# 57 x Kenyon) x CDC Bounty	Available	1000+
21	(CIMMYT # 18 x Kenyon) x HY 644	Available	1000+
22	(CIMMYT # 18 x Kenyon) x Alsan	Available	1000+
23	(CIMMYT # 18 x Kenyon) x AC Splendor	Available	1000+
24	(CIMMYT # 18 x Kenyon) x CDC Bounty	Available	1000+
25	(CIMMYT # 18 x Kenyon) x HY 475	Available	1000+
26	(*T. turgidum* # 283 x Coulter) x DT 733	Available	1000+
27	(*T. turgidum* # 283 x Coulter) x Avonlea	Available	1000+

MATERIAL and METHODS

Isolation of *Pyrenophora tritici-repentis* isolates

Diseased leaf samples, collected from common and durum wheat fields as part of the Saskatchewan Cereal Disease Survey in 2000 and 2001, plus samples collected by ourselves in 2002 and 2003 provided the material from which isolates of *P. tritici-repentis* were obtained. To recover *P. tritici-repentis* from the leaf samples, leaves were cut into 4-5 cm long pieces, washed for 2 h under cold running water to rehydrate the dry leaf and wash away large dust particles, and then surface sterilized for 30 seconds in 0.6% sodium hypochlorite solution. Subsequently, the leaf sections were rinsed in sterile distilled water and 4-5 leaves were plated on 15% water agar in 100 x 10 mm petri plates. The leaves were incubated for 24 h under continuous light at room temperature and then for 24 h in dark at 20-22°C to induce

conidiophore and conidial production, respectively. Incubated leaf pieces were observed under a stereomicroscope and conidia were picked using a sterile needle and plated on petri plates containing 15% water agar, five conidia per petri plate. The plates were incubated at 22°C for 3-4 days and portion of each fungal colony developing from a single conidium was transferred onto a petri plate containing V8P agar. After these plates had been incubated in the dark for 6-7 days at 22°C and checked microscopically to confirm that the culture was *P. tritici-repentis*, cultures were prepared for immediate use and for long-term storage. For long-term storage, 5-6 plugs from a culture were air-dried for 3-5 days in a transfer hood and placed in small sealed sterile petri dishes. These plus a V8P-agar culture of the same isolate were kept at -20°C.

Plant Material

Inoculum production, inoculation and disease rating was done as reported earlier. Race identification of each isolate was based on the disease reaction produced on a set of differential hosts consisting of hexaploid wheat genotypes Erik, Glenlea, 6B-365, 6B-662, and ND495; tetraploid wheat genotypes Coulter, 4B-242 and 4B-160; and diploid wheat line TM-441 (*T. monococcum*) (Table 13). Other cereal types, barley (cv. Harrington), rye (cv. Musketeer), oat (cv. Derby), and canaryseed (cv. CDC Maria) were also tested to determine any pathogenic variability among the isolates (Table 13). Duplicate testing of each isolate was done on a smaller differential set comprising of Erik, Glenlea, 6B-365 and 6B-662. Seeds were planted in a 4 x 4 x 4 inch pots containing Redi-Earth (W.R. Grace and Co. of Canada Ltd., Ajax, ON, Canada). One row containing 6-8 plants was seeded per pot for each of two differential genotypes.

Table 13. Disease reaction of races 1 to 5 of *P. tritici-repentis* on differential genotypes used in this study

Genotypes	Races of *P. tritici-repentis*				
	1	2	3	4	5
Erik	Resistant	Resistant	Resistant	Resistant	Resistant
Glenlea	Necrosis	Necrosis	Resistant	Resistant	Resistant
6B-365	Chlorosis	Resistant	Chlorosis	Resistant	Resistant
6B-662	Resistant	Resistant	Resistant	Resistant	Chlorosis
ND495	Necrosis	Necrosis	Resistant	Resistant	Resistant
Coulter	Necrosis	Necrosis	Necrosis	Resistant	Necrosis
4B-160	Chlorosis	Resistant	Resistant	Resistant	Resistant
TM-441	Chlorosis	Resistant	Chlorosis	Resistant	Resistant
Musketeer	Resistant	Resistant	Resistant	Resistant	Resistant
Harrington	Resistant	Resistant	Resistant	Resistant	Resistant
Derby	Resistant	Resistant	Resistant	Resistant	Resistant
CDC Maria	Resistant	Resistant	Resistant	Resistant	Resistant

RESULTS

In 2000, of 63 isolates of *P. tritici-repentis* from common wheat, 13% were identified as race 1, 84% as race 2 and 3% as race 4. Of 53 isolates from durum wheat, 21% were found to be race 1, 75% were race 2 and 4% were race 3. Isolates of race 3 were not isolated from common wheat leaf and race 4 was absent in the durum wheat leaf (Fig. 1). Similar results were observed in 2001, with 28% race 1, 69% race 2 and 3% race 4 isolates being identified from common wheat (77 isolates tested), and 36% race 1, 56% race 2 and 8% race 3 isolates identified obtained from durum wheat (64 isolates tested). In 2002, leaf samples were collected only from common wheat. Of the 64 isolates obtained, 31% were race 1, 63% were race 2 and 6% were race 4. Leaf samples from 2003 disease survey have been collected and isolation of *P. tritici-repentis* and their subsequent race designation is in progress.

All the other cereal types, barley (cv. Harrington), rye (cv. Musketeer), oat (cv. Derby), and canaryseed (cv. CDC Maria) included in the differential set for race identification gave resistant reactions to all the *P. tritici-repentis* isolates. However it was observed that canaryseed was the most resistant followed by oats, barley and rye.

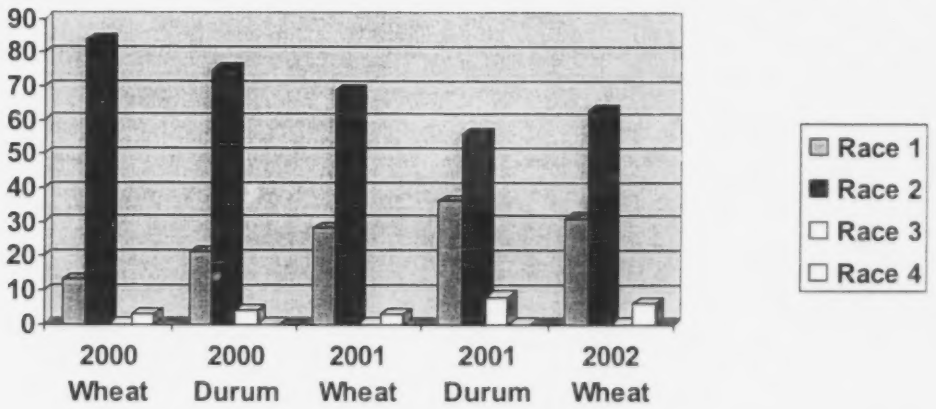

Figure 1. Percentage distribution of *P. tritici-repentis* races 1 to 4 among isolates obtained from common wheat and durum wheat, 2000-2002.

DISCUSSION

Although race 2 was the most prevalent on both common and durum wheat in the three years studied, there was an indication that its prevalence was decreasing with an increase in the occurrence of race 1. In all three years, race 3 isolates were obtained only from durum wheat and race 4 isolates were only obtained from common wheat. No isolates belonging to races 5-11 were found during this study. Without data from additional years, it is not clear whether these represent transient fluctuations or indicate a potential shift in race prevalence. If the latter is real,

studies to understand why such a shift is occurring are desirable. Since none of the present commercial cultivars widely grown in Saskatchewan possess any appreciable resistance to tan spot, host selection pressure is an unlikely factor. This suggests that environmental factors could be responsible. Fungicide application for control of tan spot has increased in recent years and could be a source of selection pressure, but it is still not a widely adopted control measure. Another possibility, given the dry conditions experienced in the years the race survey was conducted, is that *P. tritici-repentis* races differ in their response to climatic conditions; race 1 may be more adapted to spore germination and infection under drier conditions that race 2.

All the other cereal crops included in this study (barley, oats, rye and canaryseed) gave resistant reactions. This finding is in agreement with prior reports (Weise 1987; Maraite et al. 1992) which indicated that barley and oats are nonpathogenic while oats is weakly pathogenic to *P. tritici-repentis*. No prior studies have been conducted with canaryseed and under field conditions we have observed the occurrence of *P. tritici-repentis* on canaryseed (Singh et al. 2003) hence more comprehensive studies on pathogenicity of *P. tritici-repentis* on canaryseed is required.

Implications of the race survey results for breeding for tan spot resistance are clear. The two predominant races, race 1 and race 2, both produce necrotic symptoms, therefore at a minimum the gene controlling resistance to necrosis should be present in all registered wheat cultivars. Race 1 also produces chlorotic symptoms, resistance to which is controlled by a different gene, which should also be present in wheat cultivars. To identify genotypes with both resistances, race 1 should be used in tests screening for tan spot resistance. However, if the resistance genes are being incorporated separately or sequentially, a two-step screening process could be used. Use race 2 to identify resistance to necrosis, the easiest symptom to accurately identify and then test the resistant material with race 1, which would identify resistance to chlorosis plus verify the presence of resistance to necrosis.

CONCLUSIONS

1. Good sources of resistance to tan spot and stagonospora nodorum blotch of wheat have been identified. These sources show resistance to all the four virulent races of *P. tritici-repentis* identified in western Canada.

2. Genetic studies have identified that resistance to necrosis caused by *P. tritici-repentis*, race 1 is controlled by a single recessive gene. All the resistant sources identified including hexaploid and tetraploid genotypes possess the same resistance gene. It was also observed that all the newly identified resistant sources possess the same gene(s) for resistance to chlorosis caused by race 1.

3. The population of *P. tritici-repentis* revealed that races 1, 2, 3, and 4 were present in Saskatchewan. Race 2 isolates were most prevalent followed by race 1 on both durum and common wheat. Race 3 isolates were found only on durum wheat while race 4 isolates were found only on common wheat.

FUTURE RESEARCH DIRECTIONS

This study identified a number of areas in which additional research is needed if a successful strategy for control of tan spot in Saskatchewan, first by fungicides and then, as resistant cultivars become available, through host resistance.

1. Since little resistance to tan spot is present in registered wheat cultivars, fungicide control is an important control measure. However, continous use of fungicides may result in development of tolerant strains. Therefore, the ability of *P. tritici-repentis* to develop resistance or tolerance to currently used fungicides needs investigation.

2. The ability of *P. tritici-repentis* to form races, many of which appear to be controlled by different resistance genes, indicates the need to conduct an ongoing survey of the Saskatchewan population of *P. tritici-repentis* occurring on common wheat, durum wheat and wild grasses. This would monitor the race structure and indicate the appropriate resistance genes needed to develop cultivars with effective and long-lasting resistance.

3. The two resistant genes identified in these study are effective against the four races presently found in Saskatchewan. However, races 5 and 11 of P. Tritici-repentis have recently been identified in the U.S.A. The new sources of resistance identified in this study are effective against race 5 and need to be tested to determine if these sources are resistant to race 11.

4. Despite the diversity of genotypes found in this study to possess resistance to tan spot, the genetic base for resistance still remains very narrow and emphasizing that the search for additional resistance genes should continue. Since we observed resistance in wild wheat genotypes that do not possess the B genome, the location of the gene providing resistance to tan necrosis, further studies should examine the genetic basis of resistance in these wild relatives of wheat.

5. Since the newly identified resistant sources possessed resistance to both tan spot and stagonospora nodorum blotch, this suggests a study to determine if resistance to tan spot and stagonospora nodorum blotch is linked. Such a linkage would be very useful for the development of cultivars resistant to these leaf spotting diseases.

6. The barley, rye, oat and canaryseed genotypes tested were resistant to all *P. tritici-repentis* races identified by the race survey. However other studies have reported these species to be hosts of this pathogen. Therefore, further studies using optimized conditions for disease development are desirable to verify our initial finding that that *P. tritici-repentis* is non-pathogenic on these crops.

INFORMATION OF BENEFIT TO PRODUCERS AND GOVERNMENT

This study has shown that resistance to all tan spot races currently present in Saskatchewan is available for use by breeders. However, although the study identified new resistance sources, it did not identify new resistance genes. Therefore, because of the importance of this disease, particularly for durum wheat, further attempts to identify new resistance genes are needed. Similarly, ongoing monitoring of the race structure of this pathogen is needed.

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1. Singh, P.K., J. Feng, and G.R. Hughes. (2003). Race structure of *Pyrenophora tritici-repentis* in Saskatchewan Oral Presentation. Plant Pathology Society of Alberta-Canadian Phytopathological Society, Saskatchewan Meeting. October 20-22. Lloydminister, Canada.

   (Abstract to be published in Canadian Journal of Plant Pathology)

2. Singh, P.K. and G.R. Hughes. (2003). Evaluation of seedling resistance to leaf spot diseases in wheat. Oral Presentation. 74th Annual Meeting of Canadian Phytopathological Society. June 21-25. Montreal, Canada.

   (Abstract to be published in Canadian Journal of Plant Pathology)

3. Singh, P.K. and G.R. Hughes. (2003). Identification of new sources of resistance to tan spot of wheat. Oral Presentation. Soils and Crops 2003 Conference. February 17-18. Saskatoon, Canada.

   (Paper in Proceedings of Soils and Crops 2003)

Manuscripts in Submission/Preparation

1. Singh, P.K. and G.R. Hughes. Identification of new sources of resistance to tan spot and stagonospora nodorum blotch of wheat

2. Singh, P.K. and G.R. Hughes. Genetics of resistance to tan spot in durum (*Triticum turgidum*) and common wheat (*Triticum aestivum*).

3. Singh, P.K. and G.R. Hughes. Race structure of the fungus *Pyrenophora tritici-repentis* in Saskatchewan for years 2000-2002.

PERSONNEL

Pawan Singh, PDF 100% time on this project

EQUIPMENT PURCHASED

Nil

Appendix Table I. Listing of crosses made and generations produced for developing resistant breeding populations resistant to tan spot

Cross #	Cross pedigree	F_1's	F_2 Embryos	$F_{2:3}$/$F_{2:4}$/ $F_{2:5}$ Families
1	Syn. Hex. Elite # 1 x CDC Teal	20	No Embryos	Not Planted
2	Syn. Hex. Elite # 1 x Erik	30	No Embryos	Not Planted
3	Syn. Hex. Elite # 1 x Kenyon	24	No Embryos	Not Planted
4	Syn. Hex. Elite # 1 x 98W1147	50	200+ embryos	40 DH Lines
5	Syn. Hex. Elite # 9 x CDC Teal	12	200+ embryos	Not Planted
6	Syn. Hex. Elite # 9 x Erik	15	No Embryos	Not Planted
7	Syn. Hex. Elite # 9 x Kenyon	38	No Embryos	Not Planted
8	Syn. Hex. Elite # 9 x 98W1147	29	200+ embryos	110/87/81 families
9	Syn. Hex. Elite # 25 x CDC Teal	22	200+ embryos	Not Planted
10	Syn. Hex. Elite # 25 x Erik	29	No Embryos	Not Planted
11	Syn. Hex. Elite # 25 x Kenyon	28	No Embryos	Not Planted
12	Syn. Hex. Elite # 25 x 98W1147	48	200+ embryos	110/86/83 families
13	Syn. Hex. Elite # 67 x CDC Teal	16	> 50 embryos	Not Planted
14	Syn. Hex. Elite # 67 x Erik	18	> 50 embryos	Not Planted
15	Syn. Hex. Elite # 67 x Kenyon	41	> 50 embryos	Not Planted
16	Syn. Hex. Elite # 67 x 98W1147	24	100+ embryos	120/88/81 families
17	Syn. Hex. Elite # 85 x CDC Teal	19	No Embryos	Not Planted
18	Syn. Hex. Elite # 85 x Erik	2	No Embryos	Not Planted
19	Syn. Hex. Elite # 85 x Kenyon	30	No Embryos	Not Planted
20	Syn. Hex. Elite # 85 x 98W1147	27	200+ embryos	120/100/97 families
21	Syn. Hex. Elite # 85 x AC Splendor	15	Not Planted	Not Planted
22	Syn. Hex. Elite # 85 x Domain	15	Not Planted	Not Planted
23	Syn. Hex. Elite # 89 x CDC Teal	2	> 50 embryos	Not Planted
24	Syn. Hex. Elite # 89 x Erik	28	> 50 embryos	Not Planted
25	Syn. Hex. Elite # 89 x Kenyon	23	> 50 embryos	Not Planted
26	Syn. Hex. Elite # 89 x 98W1147	22	200+ embryos	120/93/86 families
27	Septoria Syn. # 57 x CDC Teal	1	No Embryos	Not Planted
28	Septoria Syn. # 57 x Erik	20	200+ embryos	Not Planted
29	Septoria Syn. # 57 x Kenyon	9	200+ embryos	120/114/112 families
30	Septoria Syn. # 57 x 98W1147	64	200+ embryos	Not Planted
31	Septoria Syn. # 57 x Crocus	74	200+ embryos	Not Planted
32	Septoria Syn. # 57 x Katepwa	32	200+ embryos	Not Planted
33	Septoria Syn. # 57 x Alsan	14	200+ embryos	Not Planted
34	Septoria Syn. # 57 x ND495	17	Not Planted	Not Planted
35	Septoria A Gen. Syn. x Crocus	18	> 50 embryos	Not Planted
36	Septoria A Gen. Syn. x 98W1147	2	No Embryos	Not Planted
37	Septoria A Gen. Syn. x ND495	14	Not Planted	Not Planted
38	92MREHTR28B x CDC Teal	1	Not Planted	Not Planted
39	92MREHTR28B x Erik	41	200+ embryos	110/83/80 families
40	92MREHTR28B x Kenyon	59	200+ embryos	110/80/80 families
41	92MREHTR28B x 98W1147	38	200+ embryos	Not Planted

Cross #	Cross pedigree	F_1's	F_2 Embryos	$F_{2:3}$/$F_{2:4}$/ $F_{2:5}$ Families
42	92MREHTR28B x Janz	24	200+ embryos	Not Planted
43	CIMMYT LINE # 18 x CDC Teal	10	200+ embryos	Not Planted
44	CIMMYT LINE # 18 x Erik	19	200+ embryos	110/86/85 families
45	CIMMYT LINE # 18 x Kenyon	31	200+ embryos	110/81/81 families
46	CIMMYT LINE # 18 x 98W1147	28	200+ embryos	Not Planted
47	CIMMYT LINE # 18 x AC Splendor	15	200+ embryos	Not Planted
48	CIMMYT LINE # 18 x Janz	3	Not Planted	Not Planted
49	CIMMYT LINE # 18 x ND495	12	200+ embryos	Not Planted
50	INTROS # 7 x CDC Teal	0	200+ embryos	Not Planted
51	INTROS # 7 x Erik	10	100+ embryos	110/94/87 families
52	INTROS # 7 x Kenyon	12	100+ embryos	110/102/96 families
53	INTROS # 7 x 98W1147	15	200+ embryos	Not Planted
54	INTROS # 7 x Domain	19	200+ embryos	Not Planted
55	2000 Spelt # 20 x CDC Teal	2	200+ embryos	Not Planted
56	2000 Spelt # 20 x Erik	16	200+ embryos	110/86/83 families
57	2000 Spelt # 20 x Kenyon	16	200+ embryos	110/98/97 families
58	2000 Spelt # 20 x 98W1147	22	200+ embryos	Not Planted
59	2000 Spelt # 20 x Domain	19	200+ embryos	Not Planted
60	2000 Spelt # 20 x AC Splendor	15	200+ embryos	Not Planted
61	2000 Spelt # 20 x ND495	22	200+ embryos	Not Planted
62	Altar*S/*Ae. squa.* x CDC Teal	1	Not Planted	Not Planted
63	Altar*S/*Ae. squa.* x Erik	8	100+ embryos	120/96/87 families
64	Altar*S/*Ae. squa.* x Kenyon	9	200+ embryos	120/111/100 families
65	Altar*S/*Ae. squa.* x 98W1147	3	200+ embryos	Not Planted
66	Altar*S/*Ae. squa.* x Janz	27	200+ embryos	Not Planted
67	Altar*S/*Ae. squa.* x Domain	8	Not Planted	Not Planted
68	Altar*S/*Ae. squa.* x ND495	28	200+ embryos	Not Planted
69	Kenyon x ND495	7	Not Planted	Not Planted
70	ND495 x Altar*S/*Ae. squa.*	17	Not Planted	Not Planted
71	ND495 x 2000 Spelt # 20	12	200+ embryos	Not Planted
72	ND495 x INTROS # 7	8	200+ embryos	Not Planted
73	ND495 x CIMMYT LINE # 18	4	Not Planted	Not Planted
74	ND495 x Erik	19	200+ embryos	Not Planted
75	ND495 x AC Splendor	11	200+ embryos	Not Planted
76	AC Splendor x 2000 Spelt # 20	26	Not Planted	Not Planted
77	AC Splendor x Altar*S/*Ae. squa.*	24	200+ embryos	Not Planted
78	AC Splendor x INTROS # 7	17	Not Planted	Not Planted
79	Janz x Kenyon	33	200+ embryos	Not Planted
80	Janz x Katepwa	35	200+ embryos	Not Planted
81	Janz x Erik	19	200+ embryos	Not Planted
82	Janz x 2000 Spelt # 20	18	Not Planted	Not Planted
83	Domain x 2000 Spelt # 20	8	Not Planted	Not Planted
84	Domain x CIMMYT LINE # 18	20	Not Planted	Not Planted

Cross #	Cross pedigree	F_1's	F_2 Embryos	$F_{2:3}$/$F_{2:4}$/ $F_{2:5}$ Families
85	*T. timopheevii* (199) x Crocus	25	No Embryos	Not Planted
86	*T. timopheevii* (199) x Katepwa	20	No Embryos	Not Planted
87	*T. timopheevii* (199) x Erik	5	No Embryos	Not Planted
88	*T. timopheevii* (199) x 98W1147	5	No Embryos	Not Planted
89	*T. timopheevii* (199) x 4B-242	19	No Embryos	Not Planted
90	*T. timopheevii* (199) x Coulter	17	No Embryos	Not Planted
91	*T. timopheevii* (199) x Avonlea	28	No Embryos	Not Planted
92	*T. dicoccides* (206) x Crocus	30	No Embryos	Not Planted
93	*T. dicoccides* (206) x Katepwa	6	No Embryos	Not Planted
94	*T. dicoccides* (206) x 98W1147	24	No Embryos	Not Planted
95	*T. dicoccides* (206) x Alsan	2	No Embryos	Not Planted
96	*T. dicoccides* (206) x Coulter	16	No Embryos	Not Planted
97	*T. dicoccides* (206) x Avonlea	9	No Embryos	Not Planted
98	*T. timopheevii* (227) x Crocus	10	No Embryos	Not Planted
99	*T. timopheevii* (227) x Katepwa	12	No Embryos	Not Planted
100	*T. timopheevii* (227) x Erik	7	No Embryos	Not Planted
101	*T. timopheevii* (227) x 98W1147	20	No Embryos	Not Planted
102	*T. timopheevii* (227) x Alsan	2	No Embryos	Not Planted
103	*T. timopheevii* (227) x Coulter	46	No Embryos	Not Planted
104	*T. timopheevii* (227) x Avonlea	14	No Embryos	Not Planted
105	*T. dicoccides* (235) x Crocus	36	No Embryos	Not Planted
106	*T. dicoccides* (235) x Katepwa	9	No Embryos	Not Planted
107	*T. dicoccides* (235) x Erik	13	No Embryos	Not Planted
108	*T. dicoccides* (235) x 98W1147	13	No Embryos	Not Planted
109	*T. dicoccides* (235) x Alsan	2	No Embryos	Not Planted
110	*T. dicoccides* (235) x 4B-242	9	No Embryos	Not Planted
111	*T. dicoccides* (235) x Coulter	7	No Embryos	Not Planted
112	*T. dicoccides* (235) x Avonlea	6	No Embryos	Not Planted
113	*T. turgidum* (283) x Crocus	16	200+ embryos	125 $F_{2:3}$ families
114	*T. turgidum* (283) x Katepwa	29	200+ embryos	Yes
115	*T. turgidum* (283) x Erik	8	200+ embryos	85 $F_{2:3}$ families
116	*T. turgidum* (283) x 98W1147	52	200+ embryos	90 $F_{2:3}$ families
117	*T. turgidum* (283) x 4B-242	20	200+ embryos	Not Planted
118	*T. turgidum* (283) x Coulter	10	200+ embryos	120/110/84 families
119	Coulter x Katepwa	16	100+ embryos	50 $F_{2:3}$ families
120	Coulter x Erik	8	No Embryos	Not Planted
121	Coulter x *T. timopheevii* (199)	8	No Embryos	Not Planted
122	Coulter x *T. dicoccoides* (235)	10	No Embryos	Not Planted
123	Coulter x *T. turgidum* (283)	9	No Embryos	Not Planted
124	Katepwa x Coulter	10	100+ embryos	50 $F_{2:3}$ families
125	Katepwa x Alsan	25	200+ embryos	Not Planted
126	Katepwa x *T. timopheevii* (227)	2	No Embryos	Not Planted
127	Katepwa x *T. dicoccoides* (235)	2	No Embryos	Not Planted

Cross #	Cross pedigree	F_1's	F_2 Embryos	$F_{2:3}$/$F_{2:4}$/ $F_{2:5}$ Families
128	Katepwa x 4B-242	14	> 50 embryos	Not Planted
129	Katepwa x *T. turgidum* (283)	2	200+ embryos	55 $F_{2:3}$ families
130	Katepwa x *T. dicoccides* (206)	1	No Embryos	Not Planted
131	Katepwa x *T. timopheevii* (199)	1	No Embryos	Not Planted
132	Crocus x *T. timopheevii* (199)	12	No Embryos	Not Planted
133	Crocus x *T. dicoccoides* (235)	6	No Embryos	Not Planted
134	Crocus x *T. dicoccides* (206)	6	No Embryos	Not Planted
135	Erik x Coulter	35	200+ embryos	Not Planted
136	Erik x *T. timopheevii* (199)	1	No Embryos	Not Planted
137	Erik x 4B-242	38	200+ embryos	30 $F_{2:3}$ families
138	Avonlea x Katepwa	2	No Embryos	Not Planted
139	Avonlea x Crocus	1	200+ embryos	Not Planted
140	Avonlea x Alsan	2	No Embryos	Not Planted
141	4B-242 x Katepwa	4	No Embryos	Not Planted
142	4B-242 x *T. timopheevii* (227)	6	No Embryos	Not Planted
143	Kenyon x 4B-242	10	200+ embryos	Not Planted